Exhibit 25

same route as MPTP and solvents i.e. oxidative stress and mitochondrial toxicity. Oxidative stress is an imbalance between the production of harmful free radicals (reactive oxygen species) and the body's ability to counteract them with antioxidants, leading to damage to brain cells. Mitochondria are membrane-bound cell organelles that generate most of the chemical energy needed to power the cell's biochemical reactions

- *Heavy metals*: Heavy metals, such as iron (Fe), mercury (Hg), manganese (Mn), copper (Cu), and lead (Pb), have been linked to PD and contribute to its progression. [22] The mechanism of cell death with heavy metals is also oxidative stress.

- *Cleaning chemicals and solvents*: A growing body of literature has demonstrated that exposure to trichloroethylene (TCE) is a risk for developing Parkinson's disease. Epidemiologic data has become more compelling over the years. The first case report of PD developing in the setting of TCE exposure (through a work exposure) was published in 1999.[24] In 2008, a cluster of TCE exposed workers had a higher incidence of Parkinson's disease than the general population. This study demonstrated that TCE was toxic to mitochondria with a mechanism similar to MPTP. [25] A twin study authored by Dr. S. Goldman published in 2011 showed that exposure to TCE increase the risk of PD over 6 fold.[26] Twin studies are particularly relevant since twins share nearly identical genetic markers but differ only in their exposure to TCE.

  Two studies examined mortality among Marine and Navy personnel and among civilian employees at Camp Lejeune, as compared to those at Camp Pendleton.[27,28] This is the first in a series of studies that compare Camp Lejeune (where TCE levels were well above the allowable level) and Camp Pendleton (where there is no evidence of TCE contamination). Mortality hazard ratios at Camp Lejeune were significantly higher for many causes, including PD. The study estimated levels of TCE and PCE in water in the Hadnot Point system during the period 1975-1985 of 359 µg/L and 16 µg/L, respectively.[27,28] The Maximum Contaminant Level (MCL) set by the EPA is 5 µg/L for TCE and PCE.[68,69]

  The next major study looking specifically at those that served at Camp Lejeune between 1975 and 1985 was published in 2023.[29] This study evaluated records of more than 150,000 veterans that served either at Camp Lejeune or Camp Pendleton between 1975 through 1985 and had a follow up between 1997-2021. The levels of TCE at Camp Lejeune were 70 times the allowable levels (deemed by the EPA) during that time frame. Results showed that those who served at least 3 months at Camp Lejeune had a 70% greater chance of developing Parkinson's disease than those that served at Camp Pendleton. This result was highly significant (P<.001). Camp Lejeune veterans also had increased symptoms of prodromal parkinsonism

suggesting that they were more likely to develop Parkinson's disease over time. Follow up of this cohort of veterans from Camp Lejeune showed that those affected by Parkinson's disease progressed more quickly with shorter time to psychosis, fracture and falls.[30] These findings suggest the PD caused by TCE may progress more rapidly.

A large study from 2024 which was a follow-up to two previous studies by Bove et al. aimed at evaluating mortality of Marines, Navy personnel and civilian workers at Camp Lejeune and at Camp Pendleton. The former were exposed to TCE and to PCE over a thirty-year period (1953-1985). The findings indicate a two-fold increase of mortality due to PD in Marines and Navy personnel at Camp Lejeune, compared to Camp Pendleton.[31] In some of my own research, we identified a cluster of attorneys with Parkinson's disease and prodromal parkinsonism who were exposed to high levels of TCE from a contaminated site near their office.[7] Their rate of PD and prodromal parkinsonism is higher than expected for age and also higher than the control cohort. A recent series of case reports from Dorsey et al included a professional athlete who spent his early childhood at Camp Lejeune and developed PD at the age of 34.[32] The above referenced studies have shown that Parkinson's disease can manifest 40-50 years after exposure to TCE but the timing of disease onset is individually variable.

In addition to epidemiologic data, animal studies have consistently shown that exposure to TCE causes damage to the dopaminergic system which mimics the changes seen in PD. The toxicity of TCE was documented in rodents of both sexes via either oral, intraperitoneal or inhalation routes of administration.[33,34] These effects are both dose and time dependent meaning the higher or longer the exposure to TCE, the greater the damage. In fact, studies show that inhalation of TCE is more toxic than ingestion due to greater dopaminergic degradation.[35] Any ingestion exposure is also associated with inhalation exposure.

Animal studies show TCE results in damage to mitochondrial function, loss of dopamine containing neurons in the substantia nigra (which is the region affected in PD), increased inflammation, accumulation of alpha-synuclein, and increased activity of LRRK2 kinase, the most common mutation associated with familial PD. These findings strongly support human studies that TCE can induce brain damage consistent with that observed in Parkinson's disease. Potential mechanisms of action of TCE induced PD are related to TCE metabolites and gut microbiome changes. TaClo is a TCE metabolite which is structurally similar to MPTP and causes similar damage to dopamine containing neurons in rodents.[36] TaClo also stimulates LRRK2 kinase activity which as noted above is the most common genetic mutation associated with PD. TCE has been shown to change the gut microbiome in the rodent model of TCE exposure which mimics the changes in gut microbiome changes in humans with PD.[37] Changes in gut microbiome are

water), (2) the duration of exposure, (3) the intensity of the exposure (as shown by the ATSDR water modeling data and other data as to the levels of the chemicals in the water) and (4) the frequency Mr. Welch was exposed in his day to day life at Camp Lejeune.

I was able to determine that Mr. Welch had substantial exposure just based upon the records at issue, Mr. Welch's deposition and the ATSDR water modeling reports. However, I additionally reviewed exposure charts provided to me from Plaintiff's expert Dr. Kelly Reynolds. Dr. Reynolds put together charts that detail a reasonable estimated dose of ingestion exposure for Mr. Welch. These charts support my opinion that Mr. Welch sustained a substantial exposure that was causally related to his Parkinson's disease. For example, Dr. Reynolds charts indicate that Mr. Welch would have likely ingested the following amounts of the toxins at issue in this case:[99]

		Chart 1: 1L	Chart 2: ATSDR	Chart 3: Deposition	Chart 4 Deposition/FM
	Cumulative ug/l-M	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
Hadnot Point					
TCE	259	6,951	8,248	10,644	13,524
PCE	0	0	0	0	0
VC	0	0	0	0	0
BZ	29	765	908	1,171	1,488
Terawa Terrace					
TCE	21	543	1,123	1,522	1,888
PCE	524	13,660	28,245	38,262	47,469
VC	28	732	1,514	2,051	2,544
BZ	0	0	0	0	0
Totals HP & TT					
TCE	280	7,494	9,371	12,166	15,412
PCE	524	13,660	28,245	38,262	47,469
VC	28	732	1,514	2,051	2,544
BZ	29	765	908	1,171	1,488

As with many diseases associated with toxin exposure, manifestation of symptoms comes years after the exposure. Mr. Welch had an approximate 11-month exposure to toxic levels of TCE while serving at Camp Lejeune between November 18, 1970 and December 15, 1971.[100] In fact, his work area was adjacent to the most highly contaminated water supply on the base (Hadnot Point) and his base apartment was supplied with contaminated base water. Epidemiologic studies outlined above have shown a statistically significant association between toxic TCE exposure and Parkinson's disease. This is further supported by extensive animal research that documents TCE exposure causing the same pathology and mechanism of cell death that occurs in humans with PD. Using the Bradford Hill framework applied by the general causation experts like Dr. Cannon, Dr. De Miranda, Dr. Miller, Dr. Costa and Dr. Boehme (strength of association, consistency, temporality, biologic gradient, plausibility, coherence experimental evidence and analogy), the

evidence strongly supports a causal relationship between TCE exposure and development of PD.

VI. Opinion on Causation

Based on my education, training, and expertise as a neurologist and a movement disorder specialist, and my review of the materials addressed and referenced in this report, and the materials listed on my reliance list as Exhibit A, to a reasonable degree of medical certainty, I conclude that Mr. Welch's Parkinson's disease is as least as likely as not due to his exposure to TCE at Camp Lejeune for an approximate 11 month period from November 18, 1970 to December 15, 1971.[100] [MOU1] [hs2]

A. Future Care Considerations

[redacted]

B. Life Expectancy Considerations

Life expectancy for those with Parkinson's disease is dependent on many factors including age of onset, progression of disease, comorbidities, etc. Studies have clarified those patients with Parkinson's disease live fewer years than age and sex matched population comparators. Men with Parkinson's disease at age 75 live on average 5 more years while those without Parkinson's disease live 10 more years.[91]